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CASE REPORT

Supplementary motor area seizures presenting as stumbling episodes

SELIM R. BENBADIS*, PRAKASH KOTAGAL*† & A. DAVID ROTHNER†

Department of Neurology, Section of Epilepsy and Sleep Disorders and Section of Pediatric Neurology†, Cleveland Clinic Foundation Cleveland, OH 44195, USA*

Address for correspondence: Selim R. Benbadis, MD, Department of Neurology, Medical College of Wisconsin, 9200 W. Wisconsin Avenue, Milwaukee, WI 53226, USA.

A healthy young boy presented with brief stumbling episodes of recent onset. Evaluation with prolonged EEG-video monitoring led to the diagnosis of supplementary motor area epilepsy. Focal seizures arising from the medial fronto-parietal cortex may present unusual diagnostic pitfalls in their clinical semeiology as well as their EEG characteristics.

Key words: seizure; focal epilepsy; diagnosis; electroencephalography; motor cortex; EEG; supplementary motor; partial seizures.

INTRODUCTION

Focal seizures arising from the supplementary motor area (SMA) have been recently described as having unusual but characteristic features[1,2]. We report a patient with an atypical presentation.

CASE REPORT

A 6-year-old right-handed boy was referred for a 2-month history of unexplained episodes. These occurred while walking, and were characterized by brief stumbling. He described that his knees would 'buckle', and this would happen once weekly to five times daily. On occasion, he would also describe a tingling sensation in both legs. He had not fallen, but nearly so.

Birth, delivery, development and past medical history were entirely unremarkable. General and neurological examinations were entirely normal. An MRI of the brain was normal. A routine EEG had shown sharp waves at the vertex.

The patient underwent 4 days of continuous EEG-video monitoring. Awake background and sleep patterns were normal. The interictal EEG showed very frequent sharp waves at the vertex during wakefulness, which occurred sporadically and in repetitive runs (Fig. 1). Seven stumbling episodes were recorded, during which the patient stumbled briefly while walking or standing. This was often subtle, but was pointed out by the patient each time. The EEG during three episodes showed no clear change from the interictal EEG. In four events, the EEG showed sharp wave discharges at the vertex, similar to those seen interictally (Fig. 2**A**,**B**), or a poorly defined rhythmic alpha also at the vertex (Fig. 2**C**); however, because of the high frequency of these sharp waves interictally, their ictal nature was uncertain. On one occasion, the patient complained of a few seconds of tingling in his left leg, during which there was no EEG correlate.

After a night of sleep deprivation, the boy experienced a different type of event. While lying in bed, his left leg flexed and he called for help ('it's happening now'). Within 2 seconds, his entire left side became stiff; he then lost consciousness and had a generalized tonic–clonic seizure. After the postictal sleepiness, he only

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Fig. 1: Interictal sharp waves, sporadic and repetitive.

recalled having felt left leg tingling. The EEG showed a rhythmic 9 Hz discharge at onset, located at the vertex, slightly to the right (Fig. 3A) which was rapidly obscured by mucsle artifact.

The child was put on carbamazepine monotherapy. The stumbling episodes have disappeared and he has been seizure-free for 6 months to date.

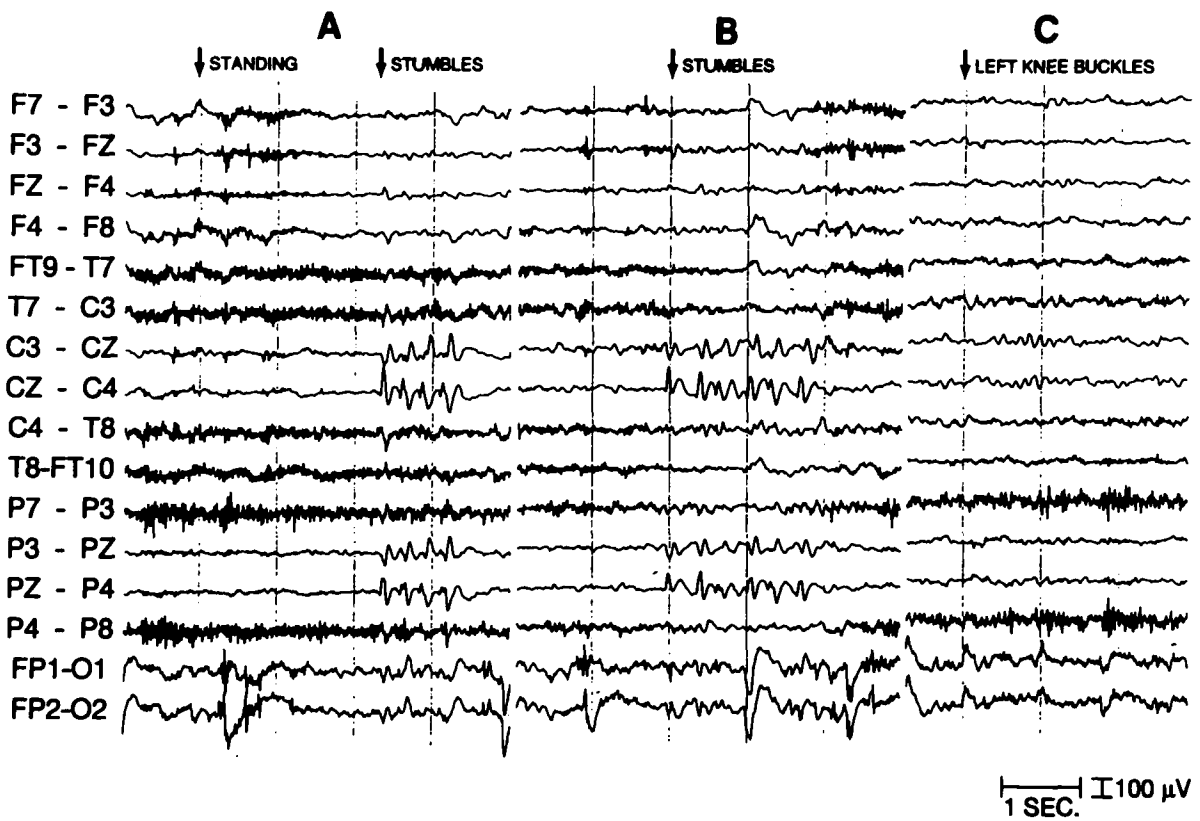

Fig. 2: Stumbling episodes; **A** and **B**, with vertex sharp waves; **C**, with rhythmic alpha at the vertex.

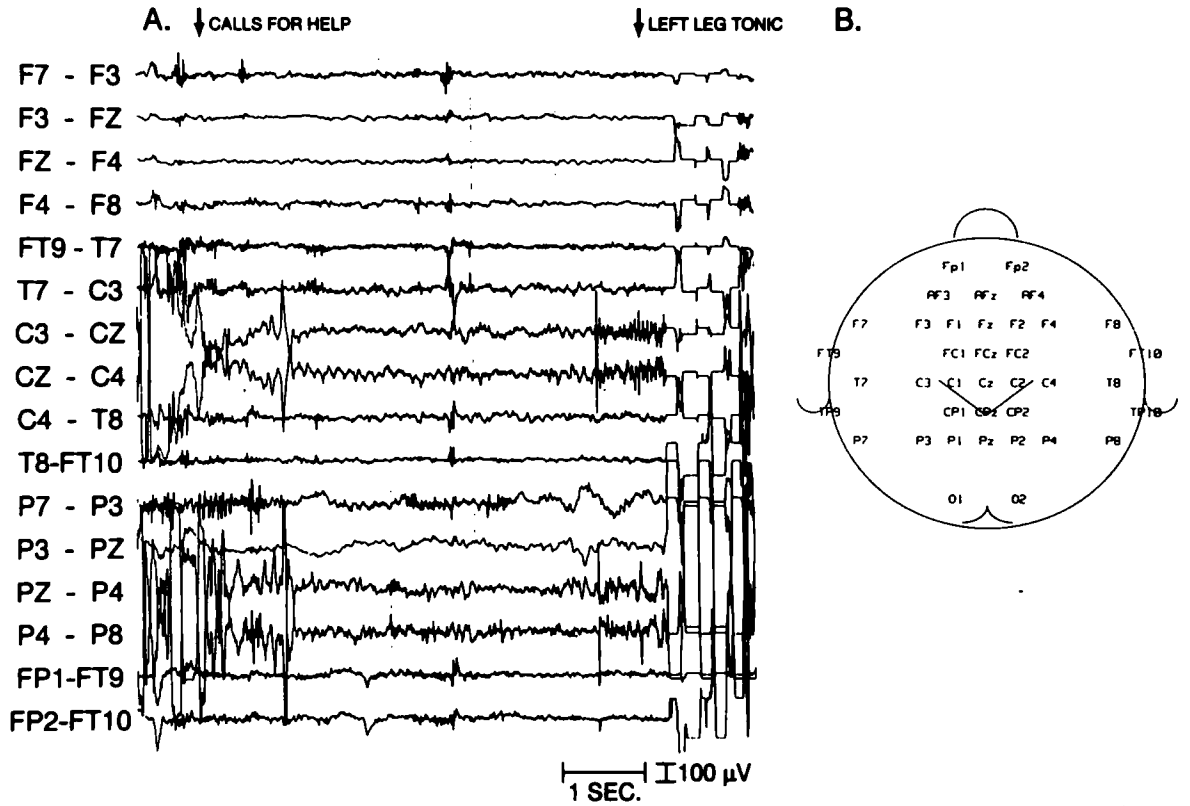

Fig. 3: **A**, EEG seizure with rhythmic 9 Hz at the vertex, rapidly obscured by artifact; **B**, Electrode placement used.

DISCUSSION

The supplementary motor area is located on the medial fronto-parietal region, and has been recognized since the work of Penfield and associates[3,4]. Electrical stimulation of this region of cortex produces motor phenomena which are distinct from those elicited by stimulation of the primary motor area[3–6]. Seizures arising from this area have only recently been described in detail[1]. They are typically characterized by tonic motor phenomena (extension or abduction) involving the proximal parts of the limbs. These, like movement produced by stimulation, may be contralateral, but also ipsilateral or bilateral. Other characteristics include short duration, preserved consciousness, and nocturnal exacerbation. Although motor phenomena are predominant in those seizures, many patients report sensory symptoms comparable to those of our patient, and indeed these can also be produced by cortical stimulation[4–6]. For those reasons, the term supplementary *sensori*motor area may be preferable to supplementary motor area (SMA)[6,7]. Like other focal seizures, SMA seizures may be secondarily generalized and may be lesional or non-lesional in aetiology.

In our patient, we recorded only one tonic seizure, which had most characteristics of SMA seizures, and was followed by secondary generalization. The exact nature of the brief stumbling episodes is unclear. They were probably 'incomplete' or 'larval', extremely brief forms of SMA seizures, possibly involving negative motor areas, some of which are located just anterior to the SMA[8]. This can only be hypothesized however, since no clear ictal discharges were seen on the EEG. Alternatively, the presence of a somatosensory aura involving the left leg may be indicative of an epileptic discharge starting in the right post-central gyrus, which later spread to involve the right SMA. In addition, the interictal and ictal EEG during that seizure were strongly suggestive of a focal epilepsy arising from the region of the vertex. This is not always the case however, and the EEG may be normal ictally as well as interictally. In the series by Morris *et al* of 11 cases[1], four had a normal interictal EEG, and four had negative ictal EEGs, either because it was normal (1 patient) or because it was obscured by muscle artifact (3 patients). Surface EEG, when positive, showed rhythmic delta, theta or alpha discharges maximal at the vertex. In our patient, the ictal EEG was rapidly obscured by EMG artifact, as illustrated in Fig. 3**A**, but an ictal pattern was seen for 3 seconds. Our patient's

interictal EEG was clearly abnormal with vertex spikes during both sleep and wakefulness. Another difficulty may arise if the midline interictal spikes are only present in sleep. In that situation, establishing their definite epileptic nature (*vs.* normal sleep transients) may be very difficult. Helpful characteristics have been previously outlined[9,10]. These include their occurrence in repetitive runs (as seen in Fig. 2), asymmetry, and a narrower field of distribution. In this respect, the use of closely spaced electrodes, as shown in Fig. 3**B**, may be helpful. Morphology may be deceiving, since extremely sharp vertex waves can be normal, especially in children. When the EEG is negative, and because of their clinical characteristics which involve bilateral motor phenomena with preserved consciousness, SMA seizures are likely to be misdiagnosed as pseudoseizures[2]. Indeed this diagnosis was entertained in our patient, and may have been made if the unequivocal tonic seizure with secondary generalization had not occurred. In that respect, sleep deprivation may have played an important role in the diagnosis of our patient.

CONCLUSION

Focal seizures with symptoms produced by activation of the SMA have unusual characteristics. This report of an atypical presentation illustrates that both the clinical semeiology and the EEG may be difficult to interpret. Because of the propensity of these seizures to resemble non-epileptic events, a high index of suspicion is necessary to make the correct diagnosis.

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